EXHIBIT 10

REPORT OF ALAN DUCATMAN, M.D.
In the case of Sullivan, et. al. v. Saint-Gobain Performance Plastics Company, No. 5:16-cv-000125-GWC (D. Vt.)

Introduction

I have been retained by the Plaintiffs as an expert in the risks, medical effects, and health effects of exposure to Perfluorooctanoic Acid (PFOA), and in the design and implementation of a clinically-appropriate program of medical monitoring for residents of Bennington and North Bennington, Vermont, who have been exposed to PFOA via their drinking water.

Qualifications

I am a Professor of Public Health at West Virginia University School of Public Health and Professor of Medicine at West Virginia University School of Medicine. I am board certified in Internal Medicine and Occupational Medicine, and have independently practiced since 1982. In the past, I have been the Chair of the Department of Community Medicine in the West Virginia University School of Medicine for many years. I also have been a member of the American Board of Preventive Medicine, which determines credentials and qualifications for board certification, and Chair of the Residency Review Committee in Preventive Medicine, which accredits clinical residency training programs. I have participated on and chaired an external scientific advising committee to the Agency for Toxic Substances and Disease Registry (ATSDR) and the National Center for Environmental Health (NEHC) of the US Centers for Disease Control and Prevention (CDC) and have a Director's Award for outstanding service to this federal agency.

Before becoming a tenured professor at West Virginia University, I was Director of the Environmental Medical Service at Massachusetts Institute of Technology (1986-1992). Before that I was an active duty Lieutenant Commander in the U.S. Navy Medical Corps, responsible

for national and international consultation concerning environmental exposures of active duty service personnel and employed civilians (1983-1986).

I have published numerous original papers, reviews, and book chapters. I have written extensively about the relationship of environmental chemicals to human disease, including, but not limited to, perfluoroalkyl substances such as PFOA. I also perform research and publish peer review articles concerning quality assurance in health care, notably in the setting of ordering and interpreting clinical testing, and increasing the value of clinical testing. I am the recipient of honors and recognitions in my field. I have been asked to provide formal and informal health science advice to leaders of organizations such as the U.S. Centers for Disease Control and Prevention, nonprofit agencies with health and safety interests including the West Virginia Bureau for Public Health, state government, and industry.

I have significant and direct experience in evaluation and medical monitoring of humans exposed to PFOA. I am familiar with the C8 Health Project, which studied the health effects of PFOA and other perfluoroalkyl substances (PFAS) on a large population that was exposed to these chemicals in the mid-Ohio Valley. I have provided advice to the leaders of that Project, and have published 17 peer-reviewed articles relating to the PFAS, mostly from the C8 Health Project data. This included a summary article (published with members of the C8 Science Panel) which described the C8 Project's design, methods, and participants. I have led a team that analyzed data from the Project, and have been responsible for a website that provided open access to summary data from the Project. I have collaborated with scientists at other institutions regarding the PFAS. My work has addressed the effects of PFOA and other PFAS on liver function and its biomarkers, thyroid function, and on serum lipid levels (including total and LDL-cholesterol), among other subjects.

My perspective is that of a clinician, with a career spanning more than three decades caring for patients and populations who have had exposures that can harm their health. My CV is attached as Exhibit 1.

Facts and Data Considered; Materials Reviewed

In preparing this report, I have reviewed materials publicly available from the Vermont Department of Health (DOH) and the Vermont Department of Environmental Conservation (DEC), including the DOH summary of events, PFOA blood test results, PFOA blood test and exposure assessment results, private well testing results, and the map of the designated areas of concern. I also have reviewed the PFOA blood test results of the named plaintiffs who consumed water contaminated with PFOA. I am familiar with the medical and scientific literature in Exhibit 2, as well as the relevant papers listed in my CV, and other medical and scientific literature concerning perfluoroalkyl substances and their health effects, and concerning medical monitoring, and the opinions that follow are based on this literature, as well as on my decades of experience with medical monitoring programs, including the C8 Health Project, and are expressed to a reasonable degree of medical certainty.

Background

PFOA is a man-made toxic substance, also known as a "toxicant." In Bennington and North Bennington (together, "Bennington), drinking water wells have been contaminated with PFOA above health standards set by the State and by EPA for a period of time. Bennington people who consumed this contaminated water have above-background levels of PFOA in their blood serum. As of January 27, 2017, the average blood serum level of PFOA among the Bennington residents tested by DOH was 10 µg/l (micrograms per liter), compared to the mean background blood serum level in the US population of 2.1 µ/l, and the highest individual blood

serum level was 1125.6 µg/l. (107) (numerical references are from Exhibit 2). The blood serum levels of PFOA measured in the plaintiffs Sullivan, Addison, Sumner, and Hausthor are 24.8 µ/l, 40.9 µ/l, 305.1 µ/l, and 204.1µ/l, respectively.

When drinking water is contaminated with PFOA, it is the experience from contamination in the mid-Ohio Valley, as well as from other sites around the world where drinking water has become contaminated, that the contamination is long-lasting and that the drinking water becomes the primary source of exposure to PFOA for those who consume it. This has been shown by the work of Emmett, Holzer, Steenland, and others. (2, 12) Consistent with the published literature, the Vermont Department of Health has found that PFOA levels in the blood of Bennington residents are strongly correlated with PFOA levels in well water. (107)

The population "half-life" of PFOA as measured in human serum has an arithmetic mean of about 3.8 years. (8, 9) We do not know the half-life of PFOA in other human organs, but we do know that PFOA is especially concentrated in the liver. (9) This means that those who drank the contaminated water in Bennington in the last 1-12 years likely have not excreted the PFOA that they have bio-accumulated in their bodies. This is also consistent with the measurement of above-background levels of PFOA in the blood serum of the exposed individuals, and the blood measures of exposure may understate the liver exposure.

In Bennington, there is established human exposure to PFOA through well water, and PFOA bio-retention in the exposed population. Thus, in Bennington, we know that exposures to PFOA above background levels have occurred, and that those exposed have retained this toxicant in their bodies.

Opinions and Bases for Opinions

Based on multiple peer-reviewed publications reporting results from exposed communities in the United States and around the world (including, but not limited to the well-

known C8 study of exposed communities in West Virginia and Ohio), and from governmental publications and assessments, we know to a reasonable degree of medical certainty that human exposure to PFOA leads to alterations in the biomarkers for and function of the liver, the thyroid, the immune system, and other organs. (1, 3, 13, 14, 15, 18, 19, 22, 28, 29, 31, 34, 35, 44) PFOA exposure is associated with excess risks of adverse health effects, as compared to the background population, and we now have clinical population outcome data on these health effects, which are summarized below. (Id.). It should be pointed out that the known associations and problems have increased over time as additional scientific work is done. This list is intended to reflect current knowledge and to be illustrative, not exhaustive:

Consistently established in multiple venues:

- Higher total and LDL ("bad") cholesterol including additional levels above treatment cut-offs; (13-17, 21-28)
- Adversely altered (higher) "liver function" tests associated with exposure; (28, 29, 30, 31, 32)
- Immune suppression including but not limited to decreased vaccine uptake; (33, 34, 35, 36, 37, 38, 39, 62, 63, 64)
- Adversely altered and abnormal uric acid (gout is more likely than not, higher uric acid is near certain); (40, 41, 42, 43)
- Endocrine disruption including but not limited to thyroid abnormalities (abnormalities of thyroid function are near certain, increased thyroid disease requiring treatment is more likely than not. Other types of endocrine disruption are more likely than not). (44 - 58)

Probable excess risk, effects with a preponderance of evidence, more likely than not, include:

- Urogenital cancers including kidney and testicular cancer;(59-61)
- Asthma; (38, 62-64)
- Developmental abnormalities including slightly lower birth weights and more markedly affected subsequent adiposity through childhood development (increased

adiposity of children at moderately increased exposure doses, with different effects at very high doses); (68 - 84, 86)

- Neurodevelopmental abnormalities including ADHD following in utero exposure; (96,97,98,99,100)
- Shorter duration of breast feeding; (65)
- Toxicant associated steatohepatitis (abbreviated as TASH, NASH, or NAFLD) as a likely explanation for several of the near certain findings. (32,66,67)

Probable excess risks needing additional investigation, include:

- Excess ulcerative colitis; (85)
- Prostate cancer; (59, 60,61, 107)
- Pregnancy induced hypertension; (87)
- Fecundity (delayed time to pregnancy) ;(72,86, 91,92,93,94,95)
- Osteoarthritis; (88,90)
- Kidney function (The direction of association is likely to be both cause and effect, and the interpretation of other outcomes are affected by kidney function. It is therefore important to collect this data as part of the biomonitoring effort regardless of causation) (42)

Adverse instances of exposures and of health effects have been documented in multiple settings of PFOA-exposure community survey data, as well as from public health registries such as NHANES in the United States and internationally. These adverse health effects pertain to a wide variety of individuals, and, to a reasonable degree of medical certainty, require the additional medical attention attained through medical monitoring for the relevant health condition and/or clinical biomarker measure. A medical monitoring program for the individuals in Bennington who have been exposed to PFOA through contaminated water should include monitoring for each of these health effects to the degree that useful testing exists. From my perspective, and based on what we know today, including the literature cited in Exhibit 2, this list would include:

A. A medical survey, based on the C8 Health Survey, and updated to include new scientific information, such as (but not limited to) diagnoses of gout and steatohepatitis (non-alcoholic fatty liver disease), as well as the birth weight and then developmental weights/heights of children. It should include a review of systems that addresses the frequency of infectious diseases and respiratory diseases, especially in children. An example of a successful questionnaire used for PFOA and other PFAS in the community setting is the C8 Health Survey Questionnaire. I consulted on the development of this questionnaire, which is available online at http://www.hsc.wvu.edu/media/4542/c8-health-project-questionnaire-v7-29-05.pdf. B.

An evaluation of relevant serum PFAS concentrations, now believed to be PFOA only. There should be a prior review of any processes and procedures that may indicate the presence of additional PFAS in drinking water so far not detected, as happened recently following application of new testing techniques in the Cape Fear, NC watershed. (See for example, https://deq.nc.gov/news/hot-topics/genx-investigation as well as https://theintercept.com/2017/06/17/new-teflon-toxin-found-in-north-carolina-drinking-water/).

C. Clinical laboratory testing of biomarkers pertinent to PFAS exposure. The clinical laboratory tests collected in C8 Health project can be viewed on line at http://www.hsc.wvu.edu/resoff/research/c8/results/clinical-laboratory-tests/
Guided by the results of this precedent population testing and current information, the following blood tests are currently proposed:

Albumin (serum)

Alkaline phosphatase (serum)

Alanine aminotransferase (ALT, formerly SGPT)

Bilirubin, direct and total

Blood urea nitrogen (BUN)

Cholesterol: total, LDL, HDL, and VLDL

C-reactive protein

Creatinine (serum)

Gamma-glutamyl trans peptidase (GGT)

Globulin (Total)

Glucose (serum)

Hemoglobin A1C (glycosylated hemoglobin)

Immunoglobulin serum concentrations of IgA, IgE, IgG, and IgM

Insulin

Nonalcoholic fatty liver disease additional marker - more sensitive markers such as cleaved cytokeratin fragments or as recommended by an expert panel, to be followed by right upper quadrant (liver) ultrasound in participants with suggestive findings and no previous explanatory diagnosis (including clinician attestation about diagnosis in medical records).

T-helper cytokines: interferon-gamma, interleukin-2, Interleukin 4

Thyroid stimulating hormone (THS). T3 uptake, and other thyroid markers as recommended by expert consensus.

Triglycerides

Uric acid [1]

[1] These tests are related primarily to liver function, immune function, kidney function, endocrine function, and lipid status. Compilation of the tests to be performed and other elements of this medical monitoring program should benefit from collaborative review and comment work, with input from peer expert reviewers from different disciplines. Such collaboration leads to better clinical biomonitoring programs and the advancement of science. (5)

This medical monitoring program should continue through a minimum 4 PFOA half-lives (about 15 years) since that is the time required for about 95% of the contaminant to be excreted. Ideally, this program would continue for the life of the participants, since feared outcomes clearly apply to the life of participants.

For these residents of Bennington, Vermont, who have been exposed to PFOA through consumption of contaminated drinking water, and have above-background levels of PFOA in their blood, these above-background levels of PFOA result in increased risks of the illnesses and adverse health conditions listed above. To a reasonable degree of medical certainty, a medical monitoring program is clinically necessary for this population to detect known PFOA-related adverse health effects as early as possible in order to minimize disease and improve health outcomes. (5) The earlier these health conditions are detected, the more effectively they can be treated.

Adequate medical monitoring for this exposed population would have two temporal components. Medical screening (initial testing) focuses on protecting the health of an exposed population, generally emphasizes early detection and response to clinical outcomes of exposure, and would include screening for the health effects and risks listed above. Medical surveillance collects prospective data on the exposed population (providing a means to follow the exposed individuals over time), and adds the potential to detect the presence of additional hazards over time, which has the potential to inform about the ongoing exposure status and to add an element of protection. For industrial chemicals, the Department of Labor, Occupational Safety and Health Administration provides support to industry and explains the role of medical monitoring for employers of workers, recognizing that industrial chemicals in the workplace can be hazardous and that one aspect of a mitigation strategy is medical monitoring.

(https://www.osha.gov/SLTC/medicalsurveillance/ accessed 6/17/17). The concepts translate to non-occupational settings when populations are exposed to industrial chemicals.

A typical medical surveillance program collects survey questionnaire demographic information describing the participants, survey questionnaire health information directed at the population needs, as well as laboratory and/or radiographic information pertinent to detection of known exposure risks. Depending on the nature of exposure, the survey may collect targeted health information from the participants, as this can inform the data collected and seamlessly can be turned into de-identified community summary data. Medical surveillance may also collect simple physical examination information such as height, weight, and blood pressure. There usually is a centralized data base so that meaningful reporting of summary data and key findings can occur.

Other important benefits to uniform data storage include the ability to provide personal materials back to properly-identified participants who have lost their original data (in the C8 Health Project, participants were provided their personal data, and there is a means for participants to identify themselves and obtain their data again). It is also very helpful to the members of the affected community to have an accessible health communications strategy that provides information and processes, procedures, and summary findings for the benefit of the community, but available to anyone with internet access. This should be transparent, accessible to anyone, and have no means to identify individuals. The point about identification is important. In the C8 Health Study, focus group interviews of potential participants repeatedly mentioned loss of privacy as their largest fear. Any process which threatens loss of privacy is problematic. In that regard, the federal government provides guidance about the public reporting of rare diseases in small populations, with helpful guidelines for protecting privacy.

The truism that individuals are unique is an argument **for** an appropriate medical monitoring program for the exposed population, rather than against, or a determinant of who should be included. In general, the uniqueness of individuals is understood in the medical and public health communities to illustrate the need to monitor more of the exposed population, not less. That people have varying medical histories, medical co-morbidities, and inherited genetics means that we are aware of excess risks to the exposed population, but generally are unable to predict in advance who in the population with demonstrable excess risk will get which disease known or suspected to be associated with exposure, or when. In addition, we cannot predict the severity of the health effects for any particular individual. There are no known medical co-morbidities that mitigate the risks from PFOA exposure. Rather, we must work to identify co-morbidities that may further increase these risks.

Consider the half-life established for PFOA, by Olsen and colleagues from 3M, is a population mean, because the individuals tested did not all have the same rates of excretion. The data are valuable despite the differences among those who were tested. (8) In health care, health professionals in many organizations collect data and become increasingly expert at predicting average tendencies, including factors that bring increased risk, yet we remain persistently mediocre or worse at predicting who among the high risk community specifically will be the one(s) to suffer the outcome(s). This necessitates monitoring all of an exposed population, and is why children are monitored for lead exposure in communities where that exposure is possible.

Individual medical records and histories are irrelevant to whether a medical monitoring program, as described above, is clinically necessary for exposed Bennington residents. While individualized factors, such as age, sex, height, weight, prior medical conditions, and a host of

other differences among/between all people, can and do inform data collection, this information can be collected most efficiently and effectively though an appropriate survey, as part of the medical monitoring program.

For example, we screen a small subset of adults for lead poisoning -- workers with potential lead exposure -- because we generally can target those as being most at risk. We screen many thousands of young children, including those who live in areas with different risks of lead poisoning, because the exposure is even more serious in children, and the behaviors that lead to childhood exposure are highly individualized and hard to predict. Further, we know that some conditions, such as inadequate diet, may increase the risk of childhood lead poisoning, but this does not dissuade us from monitoring, or limit those whom we monitor.

Similarly, we do not limit monitoring based on more than one source of exposure. We know that workers in a benzene monitoring program may have exposure from two (or more) sources, such as work and personal cigarette smoking, but that supports the need to include all workers, smoking and nonsmoking, in the monitoring program, and to pair demographic data collected in a worker survey to the laboratory results. We also know that some of the risks of asbestos exposure are potentiated by cigarette smoking (such as lung cancer and parenchymal asbestosis), whereas other diseases (such as malignant mesothelioma) are related to asbestos exposure and not potentiated by smoking. Smoking status is determined in surveys, but the status does not determine eligibility for monitoring in affected communities. It is also why demographic and independent risk data are collected in the survey as part of the program which collects health and exposure data from the participants rather than trying to extract it from medical records. It should be noted that no one in Bennington has a current, formal diagnosis of

a PFAS-related disease that would exclude participation. When such formal declarations are made, the exclusions can be added.

In clinical care, if a risk is known to exist, we monitor and evaluate that risk. In Bennington, multiple adverse health risks are present in the exposed population as a direct result of their exposure to PFOA in drinking water, and for this reason they should be monitored, irrespective of individual differences. The Bennington population is homogenous only in their exposure to PFOA through their drinking water, and in their resulting increased risks of adverse health outcomes and abnormal biomarkers of various body system functions. These homogeneities determine the medical necessity of an appropriate medical monitoring program, just as exposure to asbestos or to lead determines the necessity of participation in an asbestos or lead medical monitoring program. Individualized predictions of outcomes are not possible, nor are they beneficial to the exposed population, because we do not know enough to make such individualized assessments. But, we do know that every exposed person faces increased risks of related adverse health effects.

The negative consequences of requiring further homogeneity among the exposed population, or individualized determinations of who among this exposed population should be included in the monitoring program, would be at least twofold. First, the cost of the monitoring would increase, because the review of medical records for inclusion/exclusion criteria is more expensive and far less targeted than gathering the same information through a participant survey. Second, the goal of such an effort would be to exclude members of the exposed population. Exclusion of these exposed individuals might carry health impacts to them, and would decrease the effectiveness of the program for data collection and advancing medical and scientific knowledge concerning the health effects of exposure to PFOA.

The possibility that members of the exposed population may have been exposed to PFOA through other means does not diminish the clinical necessity of medical monitoring for the exposed population. First, the literature establishes that drinking water contaminated with PFOA is the primary source of exposure for those who consume it, and, consistent with this literature, the Vermont Department of Health has found a strong correlation between the PFOA levels in the blood of Bennington residents and the PFOA levels in well water, a finding fully compatible with and predicted by existing peer literature. (2, 12, 107) Second, possible subsidiary sources of PFOA – to the extent they exist - do not impact the clinical necessity of medical monitoring based on this primary exposure. Subsidiary sources are expected and documented in predecessor efforts for populations who have consumed contaminated water and do not negate the findings about primary sources in numerous studies including but not at all limited to the population investigated in the C8 Health Study. (2, 12, 104, 105) When drinking water is not contaminated, however, community exposures are from other sources, and, biological burdens are lower. (106) The exposure through contaminated drinking water and documented levels of PFOA in blood serum establish the foregoing health risks and justify a medical monitoring program for the exposed population in Bennington. Any subsidiary sources of exposure support the collection of additional exposure data in the survey history, rather than impacting the clinical need for medical monitoring of the exposed population.

Similarly, the possibility that Bennington residents exposed to PFOA in their drinking water may already have had one or more of the adverse health conditions linked to PFOA exposure, or may have other risk factors for one or more of these adverse conditions (such as smoking or obesity), does not argue against the creation of a medical monitoring program for the entire exposed population. PFOA exposure is known to cause increased health risks to the entire

group of people exposed, regardless of their medical state, and some may be more vulnerable because of their medical state. No one has been declared to have a PFOA associated condition by any formal body, and those how may have outcomes known to be associated with exposure need not have just one such outcome. Further, until biological burdens are measured, ecologic data must substitute for biological burden data. To the extent individuals within the exposed population have a pre-existing reason for a particular test or monitoring measure that is independent of their PFOA exposure, that is not a justification for denying them access to the monitoring program necessitated by their PFOA exposure. To the contrary, increased preexisting risk is a reason to increase our attention to medical monitoring.

Let's return to the example of medical monitoring of children in communities for lead poisoning. The proper role for gathering individualized details concerning the monitoring program participants is planned as part of the medical monitoring program. When a disease state is detected, such as lead poisoning, additional medical (and other) data are collected after the program detects the poisoning, when this subsequent information is targeted to need, and can inform the administration of the program, the treatment of the participants, and the ongoing generation of data and research.

In my career, I have participated directly in the following medical monitoring projects:

- the C8 Health Study, which began in 2005 and now is in its second phase. I consulted in the choice of the initial phase laboratory tests, and in the design of the initial phase study questionnaire;
- the U.S. Navy Asbestos Medical Surveillance Program, which involved many thousands of Department of Defense employees, during my active duty as a Lieutenant Commander in the U.S. Navy Medical Corps from 1983-1986;

- Medical surveillance programs, including surveillance for lead poisoning, at the Massachusetts Institute of Technology, where I was Director of the Environmental Medical Service from 1986-1992;
- the West Virginia Lead Poisoning Prevention Program, as a consultant for program design and for patients determined to have lead poisoning, from its creation in around 1992-present;
- multiple medical surveillance programs for workers at West Virginia University, from 1992-present.

In all of these projects and programs, admission to the program was based solely on exposure to the relevant toxic substance or hazard (PFOA, asbestos, lead, noise, etc.) and the risks resulting from that exposure, and was available to entire exposed population. None of these projects and programs involved or included pre-admission review of medical records, and none was based on individualized determinations of who among the population considered to be exposed should be excluded or participate. The programs were based on the potential for exposure.

Further, all clinical interventions require consideration of potential harm, but this provides no basis for denying or individualizing the medical monitoring program for Bennington residents exposed to PFOA. Potential issues relating to harms can and should be dealt with through strong health communications and informed choices. They are not sufficient reasons for narrowing those eligible for monitoring where there exists excess risks and appropriate tests responsive to these risks. In the C8 Health Study, we have random participant survey evidence that participants appreciated the experience, and some participants sought and received further care from their doctors as a result. The C8 Health experience is that people exposed to PFOA

overwhelmingly want to understand their exposure and potentially-associated health outcomes, and are understandably anxious to learn more, rather than less. Giving primacy to a concern that they will misunderstand or misuse their data creates, rather than relieves, anxiety and legitimate concerns over health.

Moreover, individualized review of medical records, and individualized design of a monitoring program, are time-consuming, expensive, and inefficient. Many participants in the program will have normal test results and biomarkers, and review of their medical records would serve no medical purpose. In the monitoring programs listed above, individualized consideration and, if needed, record review, was employed only for those whose tests were abnormal, as part of insuring appropriate investigation of those results, and, if appropriate, further treatment. Similarly, individualized design of testing protocols would make the Bennington program far more expensive and less efficient. I have not participated in a medical monitoring program in which we attempted to sculpt the program elements to each individual based on prior review and calculation.

From a medical perspective, another consideration is the protection of the participants' privacy and confidential medical information. In the C8 Study, focus groups of the exposed population repeatedly expressed concern over whether their confidential information would be available to the company that was responsible for their exposure, and many would not have participated in the monitoring program absent firm protocols protecting their privacy and confidential medical information.

For all of these reasons, I reject any suggestion that individual uniqueness is an argument against a common medical monitoring program for the Bennington residents who have been exposed to PFOA in their drinking water, or that individual histories or records are relevant to

determining the clinical appropriateness of medical monitoring, or who among the exposed population should be included in the program. Doctors reject the idea that individual uniqueness is an argument against the usefulness of evidence-based testing and evaluation in any clinical setting.

Prior Testimony

A list of other cases in the last four years in which I have testified as an expert at trial or at deposition is attached as Exhibit 3.

Compensation

West Virginia University charges $600 per hour for my time in preparing this Report.

This the 1st day of September, 2017.

Alan Ducatman, M.D.